



**INCIDENCE OF
CENTRAL VESTIBULAR DYSFUNCTION
Data base study**

**Protocol for partial fulfillment of Master Degree in
OTORHINOLARYNGOLOGY.**

BY

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List of tables

Table number	Title
Table ١,١	Causes of central vertigo
Table ١,٢	Comparison of Posterior Inferior Cerebellar Artery (PICA) and Anterior Inferior Cerebellar Artery (AICA) Syndromes. (Cass et al; ١٩٩٩)
Table ١,٣	Central versus peripheral vertigo (Baloh; ٢٠١١)
Table ٣,١	Analysis of dizzy patients by history
Table ٣,٢	Analysis of dizzy patients by examination.
Table ٣,٣	Analysis of dizzy patients by VNG.
Table ٣,٤	Distribution of central vertigo

Abbreviation

AVS	Acute vestibular syndrome
AICA	Anterior inferior cerebellar artery
BPPV	Benign paroxysmal positional vertigo
BB	Beta Blockers
CNS	Central nervous system
CT	Computed tomography
EMG	Electromyography
ENG	Electronystagmography
HINTS	Head-Impulse-Nystagmus-Test-of-Skew
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
MV	Migrainous vertigo Migraine vestibulopathy
MS	Multiple sclerosis

PAN	Positional alcohol nystagmus
SSS	Subclavian steal syndrome
TIAs	Transient ischemic attacks
VA	Vertebral artery
VAST	Vertebral artery screening test
VB	Vertebrobasilar
VBI	Vertebrobasilar insufficiency
VM	Vestibular migraine
VR	Vestibular rehabilitation
VNG	Vestibulonystagmuography

ACKNOWLEDGMENT

- ALL THANKS TO ALLAH -

*I wish to express my deepest gratitude and thanks to **Prof. Dr. Badr El-Din Mostafa** for his help and expert supervision.*

He has been very kind, generous with scientific advice and a strong motivating force to produce an accurate research for him I will remain humbly grateful

*I wish to express my sincere gratitude to **Prof. Dr. Ayman Mohamed El-Kahky** for his continuous help, cooperation and encouragement.*

*I wish also to express my deep appreciation to **Dr. Hesham Abdl-Aty Abdl-Kader** for his close supervision and honest assistance.*

I would like to thank my family and my friends as it would not have been possible to conduct this study without their blessings and patience and to the soul of my lovely father I dedicate this work.

Last, but not least, I must express my deepest thanks to all persons who helped me in this work .

Introduction

One of the most common complaints in clinical practice is dizziness. (*Jász, et al; 2017*). Dizziness can be explained by the disturbed sense of relationship to space and also the painless head discomfort. It has many causes like disturbance in vision, the brain, and the vestibular system.

The role of otolaryngologist includes clarifying the subset of patients who have vertigo and differentiating central from peripheral vestibular disorders.

The most common causes of peripheral vestibular dysfunction are benign paroxysmal positional vertigo, vestibular neuritis, and Meniere's syndrome (*Karatas; 2017*).

The presence of other neurological abnormalities leads to an investigation for a central cause of the vertigo. However central vestibular disorder due to a lesion or stroke may mimic peripheral vestibular disorders. The goal of this work is to identify the incidence of central vestibular dysfunction among the patient presenting to the vestibular unit in our department.

Review of literature

Dizziness

Dizziness can be classified into four major types: (١)Vertigo,(٢)Disequilibrium,(٣) Lightheadedness, (٤) Presyncope and Syncope (**Rosenberg et al; ٢٠٠٠**).Vertigo can be described as an unreal sense of movement (**Fetter; ٢٠٠٠**).

The value and function of the vestibular system may often be underestimated when considering the various special senses that we possess. However, of all the special senses, loss of vestibular function may cause the most significant determinant for our daily function and survival.

Injury to the peripheral and central vestibular system causes asymmetry in the baseline input into the vestibular centers and this causes vertigo and nystagmus (**Walker et al; ٢٠٠٠**).

The duration, the periodicity, and the circumstance of the vertigo and the presence of other neurological and auditory signs or symptoms allow for categorization of vertigo into central and peripheral one.

Although the epidemiology of vertigo and vestibular disorders is still an underdeveloped field. Recent studies have underscored the impact of vertigo at the population level, but its determinants and outcome are not well known yet (**Neuhauser, ٢٠٠٧**).

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Central vestibular dysfunction or vertigo due to neurological or systemic origin is a relatively common complaint making up to ۲۰٪ of patients coming to the vestibular clinics (**Shepard; ۲۰۰۵**). Younger populations are commonly affected by migraine, headaches and multiple sclerosis. Old age groups are typically affected due to the associated risk factors of vascular causes of vertigo such as hypertension, atherosclerosis, and diabetes mellitus. Central causes of vertigo result from either: Disruption of central integrators (brainstem, cerebellum) or sensory information mismatch (cortex).

Lesions affecting vestibular nerve or root entry zone (Cerebellopontine angle lesions) result in imbalance by affecting primary vestibular sensory information.

Most common causes of central vestibular dysfunction can be classified to:

I- Vascular:
<ul style="list-style-type: none"> * Stroke. * Migraine * Vertebrobasilar insufficiency * Subclavian steal syndrome. * Lateral medullar syndrome. * Vasculitis. * Hematological
II- Epilepsy
III- Drugs e.g.: Digitalis and hypotensive drugs.
IV- Tumors and trauma to the brain.

V- Infection.
VI- Multiple sclerosis.
VII- Psychological.

Table 1.1 : Causes of central vertigo

Migrainous vertigo

Migrainous vertigo (MV) is a vestibular syndrome caused by migraine and presents with attacks of spontaneous or positional vertigo lasting seconds to days and migrainous symptoms during the attack and distinct from basilar type migraine (**Neuhauser & Lempert ; 2004**).

Migraine and vertigo are common disorders, affecting about 14% and 10%, respectively, of the general population. (**Lempert et al; 2009**).

Concurrence of the two conditions can be expected in about 1.1 % of the general population by chance alone. However, recent epidemiological evidence suggests that the actual co morbidity is higher, namely 3.2 %. This can be explained by the fact that several dizziness and vertigo syndromes occur more frequently in migraineurs. In addition, there is increasing recognition of a syndrome called vestibular migraine (VM), which is vertigo directly caused by migraine. VM affects more than 1 % of the general population.

The pathogenesis of VM is uncertain, but migraine mechanisms may interfere with the vestibular system at the labyrinth, brainstem, and cerebral cortex (**Daroff et al, 2009**).

Localized vertebrobasilar vasoconstriction leading to transient posterior circulation ischemia which may contribute to the symptomatology of the disorder. Dizziness may also be due to orthostatic hypotension, anxiety disorders or major depression

which all has an increased prevalence in patients with migraine (**Lempert et al; ٢٠٠٩**).

VM does not fit into the ٢٠٠٤ International Headache Society Classification, in which "basilar-type migraine" must have at least two posterior circulation manifestations.

MV being the most common cause of spontaneous (nonpositional) episodic vertigo. (**Eggers; ٢٠٠٦**)

VM presents with attacks of spontaneous or positional vertigo lasting seconds to days. Migrainous accompaniments such as headache, phonophobia, photophobia or auras are common but not mandatory. Cochlear symptoms may be associated but are mostly mild and non-progressive. During acute attacks one may find central spontaneous or positional nystagmus and, less commonly, unilateral vestibular hypofunction. In the symptom-free interval, vestibular testing adds little to the diagnosis as findings are mostly minor and non-specific.

Treatment includes vestibular suppressants for acute attacks and migraine prophylaxis for patients with frequent recurrences. Abortive medications such as triptans are effective for migraine related dizziness whether with headache or not. However, treatment efficacy has not been validated by properly controlled clinical trials.

Vertebrobasilar insufficiency

Vertebrobasilar insufficiency or transient ischemic attacks (TIAs) is a state of hypo perfusion to brain stem and cerebellum due to stenosis and/or spasm of the vertebral or basilar artery.

Approximately one fourth of strokes and TIAs occur in the vertebrobasilar distribution in old ages.

Causes includes: Atherosclerosis (the most common cause). Vertebral artery (VA) stenosis caused mainly by atherosclerosis accounts for up to ۲۰% of posterior circulation strokes. Congenital or skeletal compression like cervical spondylosis (**Moubayed & Saliba; ۲۰۰۹**).

Vertigo is the most common symptom of transient ischemia within the vertebrobasilar system. Episodes typically come on abruptly and last minutes (**Baloh; ۱۹۹۵**).

There are usually associated symptoms like: diplopia, facial numbness, dysarthria, headache and syncope lastly.

Based on neuroanatomical findings, these short-lasting vertigo attacks result from transient insufficiency of the anterior inferior cerebellar artery (AICA), which supplies the inner ear and leads to ischemia of the upper parts of the vestibular labyrinth (**Berlit; ۱۹۹۸**).

Persisting vertigo with or without lateropulsion may occur in cerebellar infarctions with involvement of the medial inferior hemispheres due to occlusion of the medial branch of the posterior inferior cerebellar artery.

Caloric testing with oculography can usually differentiate cerebellar infarction and peripheral vestibular disease. Since ischemic lesions with AICA insufficiency may cause pathological results in caloric testing, both clinical and neurophysiological analysis of associated oculomotor signs is essential for a correct diagnosis. MRI and MRA are hallmark in detecting occlusions and stenosis of vertebrobasilar circulation and far more sensitive to detect small ischemic foci that characterize branch occlusion.

The findings of recurrent isolated vertigo and bilateral vestibular hypo function should not prevent a search for vertebrobasilar ischemia, particularly in the presence of vascular risk factors. (**Lee et al; ۲۰۱۱**).

Multiple sclerosis

Multiple sclerosis is a chronic degenerative disorder that affects nerve fibers in the brain and spinal cord randomly in time and space.

Multiple sclerosis (MS) may give rise to a variety of clinical signs and symptoms including vertigo and/or other problems related with equilibrium (**Ardic et al; ۲۰۱۰**). Vertigo reported in some cases as main symptoms of multiple scelrosis.

Approximately ۱ per ۱,۰۰۰,۰۰۰ people acquires MS (**Bir et al; ۲۰۱۰**), the average age of onset is between ۱۸ and ۳۵ but it may develop at any age.

The exact cause of MS is unknown and some classified it as autoimmune disease.

MS characterized by intermittent damage to myelin (demyelination) caused by destruction of the oligodendrocytes that form the myelin.

MS is classified according to frequency and severity of neurological symptoms and the ability of the CNS to recover into:

- ۱- Primary progressive MS.
- ۲- Relapsing-Remitting MS (Inactive).
- ۳- Secondary progressive Ms.
- ۴- Relapsing progressive MS.

Common multiple sclerosis symptoms include fatigue and weakness; decreased balance, spasticity and gait problems; depression and cognitive issues; bladder, bowel, and sexual deficits; visual and sensory loss; and neuropathic pain. Less-common symptoms include dysarthria and dysphagia, vertigo, and tremors. Rare symptoms in multiple sclerosis include seizures, hearing loss, and paralysis. (**Ben-Zacharia, ۲۰۱۱**)

Vertigo in multiple sclerosis is mimicking benign paroxysmal positional vertigo with acute onset in diseased patients.

MRI is mandatory for lesion detection. (**Varaki et al;** ٢٠٠٨).

The vestibular rehabilitation (VR) exercises are useful in reducing complaints of MS associated vertigo beside the medical treatment of MS (**Zeigelboim et al;** ٢٠١٠).

Hyperventilation syndrome

Hyperventilation is more rapid and deep breathing than normal which its actual cause is unknown and may be due to anxiety or severe pain.

The overall incidence of Hyperventilation-induced nystagmus was ٢١,٩%. It was detected more frequently in retro cochlear vestibular diseases rather than in end-organ vestibular diseases. (**Vassallo et al;** ٢٠١١).

Hyperventilation patients rely when stressed in thoracic breathing rather than diaphragmatic breathing resulting in a hyper expanded chest and high residual lung volume that make them unable to take normal tidal volume with consequently dyspnea.

Subclavian steal syndrome

The subclavian steal syndrome (SSS) refers to a vascular disorder in which occlusion or stenosis of the subclavian artery proximal to the vertebral artery origin causes altered vascular haemodynamics that result in retrograde blood flow in the ipsilateral vertebral artery toward the upper arm, distal to the subclavian artery narrowing, where decreased blood pressure had been established.

The rate of SSS is estimated at ١,٣% in patients referred for carotid and vertebral artery Doppler ultrasound, most frequently in

Caucasians because of the increased incidence of atherosclerosis in this population. SSS generally occurs in patients > 60 years of age and has a 1:1 male-to-female ratio. (**Perler & Becker; 1998**).

Subclavian artery lesions are usually asymptomatic because of the abundant collateral blood supply in the head, neck, and shoulder. However, these lesions produce neurologic symptoms when compensatory flow to the subclavian artery from the vertebral artery diverts too much flow toward the arm and away from intra-cranial structures leading to vertebrobasilar insufficiency (VBI)

Clinically, SSS may be suspected when the difference in blood pressure between the two arms exceeds 20 mmHg, but the diagnosis is essentially confirmed by Doppler ultrasound, Magnetic resonance imaging (MRI) with or without magnetic resonance angiography (MRA), computed tomography (CT) scan of the brain and digital subtraction angiography can also be used (**Henry; 2004**).

As the vertebrobasilar arterial system feeds both the peripheral and central auditory and vestibular systems, in subclavian steal syndrome, neurotological symptoms are expected because of the vertebrobasilar insufficiency.

The otological symptoms presented with isolated dizziness, recurrent vertigo, hearing loss and tinnitus. A positional nystagmus was also detected.

Abnormal saccades and reduced caloric response is detected in patients with SSS.

These findings suggest that the central auditory and vestibular system is more likely to be involved in the pathogenesis of neurotological symptoms in subclavian steal syndrome (**Baloh; 2001**).

Cerebrovascular disease

It is any interruption of the blood supply to any part of the brain associated with a sudden loss of brain function.

Stroke is the third most common cause of death and an important cause of disability in adults.

It may be due to: ischemia (80%), thrombosis, hemorrhage (20%), and embolism.

Acute audiovestibular loss is a common neurotological condition that is characterized by sudden onset of severe prolonged (lasting days) vertigo and hearing loss and is diagnosed by the presence of canal paresis to caloric stimulation and sensorineural hearing loss on pure tone audiogram (Lee; 2012).

Acute audiovestibular loss is an important sign for the diagnosis of infarction in some territory of the brain.

Audiovestibular loss may serve as an alarming sign to prevent the progression of acute audiovestibular loss into more widespread areas of infarction in posterior circulation (mainly in the AICA territory). We should keep in mind that acute audiovestibular loss may herald impending AICA territory infarction, especially when patients had basilar artery occlusive disease, even if other central signs are absent and MRI does not demonstrate acute infarction (Lee; 2012).

Skew predicts brainstem involvement in acute vestibular syndrome (AVS) and can identify stroke when an abnormal horizontal head impulse test falsely suggests a peripheral lesion. A 3-step bedside oculomotor examination (HINTS: Head-Impulse-Nystagmus-Test-of-Skew) appears more sensitive for stroke than early MRI in AVS (Kattah et al; 2014).